```
RELATED SEQUENCES AVAILABLE WITH SEQLINK**

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
               2 REFERENCES IN FILE CA (1907 TO DATE)
               2 REFERENCES IN FILE CAPLUS (1907 TO DATE)


=> E phenylethanolamine N methyltransferase
E1          588     PHENYLETHANOL/BI
E2           45     PHENYLETHANOLAMINE/BI
E3            0 --> PHENYLETHANOLAMINE N METHYLTRANSFERASE/BI
E4            2     PHENYLETHANOLATE/BI
E5            6     PHENYLETHANOLATO/BI
E6          307     PHENYLETHANON/BI
E7          306     PHENYLETHANONATO/BI
E8         2582     PHENYLETHANONE/BI
E9           53     PHENYLETHANOYL/BI
E10           6     PHENYLETHANOYLAMINO/BI
E11           1     PHENYLETHANOYLOXY/BI
E12           1     PHENYLETHANYL/BI

=> s E3
            45 "PHENYLETHANOLAMINE"/BI
       6985205 "N"/BI
         13030 "METHYLTRANSFERASE"/BI
L6           9 "PHENYLETHANOLAMINE N METHYLTRANSFERASE"/BI
                 (("PHENYLETHANOLAMINE"(W)"N"(W)"METHYLTRANSFERASE")/BI)

=> d

L6   ANSWER 1 OF 9  REGISTRY  COPYRIGHT 2006 ACS on STN
RN   479920-94-0  REGISTRY
ED   Entered STN:  22 Jan 2003
CN   Phenylethanolamine N-methyltransferase (human clone MGC:34570
     IMAGE:5175571) (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   GenBank AAH37246
CN   GenBank AAH37246 (Translated from: GenBank BC037246)
FS   PROTEIN SEQUENCE
MF   Unspecified
CI   MAN
SR   GenBank
LC   STN Files:   CA, CAPLUS

**RELATED SEQUENCES AVAILABLE WITH SEQLINK**

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
               2 REFERENCES IN FILE CA (1907 TO DATE)
               2 REFERENCES IN FILE CAPLUS (1907 TO DATE)


=> d L6

L6   ANSWER 1 OF 9  REGISTRY  COPYRIGHT 2006 ACS on STN
RN   479920-94-0  REGISTRY
ED   Entered STN:  22 Jan 2003
CN   Phenylethanolamine N-methyltransferase (human clone MGC:34570
     IMAGE:5175571) (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   GenBank AAH37246
CN   GenBank AAH37246 (Translated from: GenBank BC037246)
```

```
FS   PROTEIN SEQUENCE
MF   Unspecified
CI   MAN
SR   GenBank
LC   STN Files:   CA, CAPLUS

**RELATED SEQUENCES AVAILABLE WITH SEQLINK**

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
               2 REFERENCES IN FILE CA (1907 TO DATE)
               2 REFERENCES IN FILE CAPLUS (1907 TO DATE)


=> s L6
            45 "PHENYLETHANOLAMINE"/BI
       6985205 "N"/BI
         13030 "METHYLTRANSFERASE"/BI
L7           9 "PHENYLETHANOLAMINE N METHYLTRANSFERASE"/BI
                 (("PHENYLETHANOLAMINE"(W)"N"(W)"METHYLTRANSFERASE")/BI)

=> File CAPLUS
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                    52.50     227.41

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                       ENTRY    SESSION
CA SUBSCRIBER PRICE                                     0.00      -8.96

FILE 'CAPLUS' ENTERED AT 12:29:39 ON 11 SEP 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)


Copyright of the articles to which records in this database refer is
held by the publishers listed in the PUBLISHER (PB) field (available
for records published or updated in Chemical Abstracts after December
26, 1996), unless otherwise indicated in the original publications.
The CA Lexicon is the copyrighted intellectual property of the
American Chemical Society and is provided to assist you in searching
databases on STN.  Any dissemination, distribution, copying, or storing
of this information, without the prior written consent of CAS, is
strictly prohibited.

FILE COVERS 1907 - 11 Sep 2006  VOL 145 ISS 12
FILE LAST UPDATED: 10 Sep 2006  (20060910/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply.
They are available for your review at:

http://www.cas.org/infopolicy.html

=> s L6
L8          1248 L6

=> s L8 and (polymorphism or mutation or allelic)
          132926 POLYMORPHISM
           38510 POLYMORPHISMS
          138975 POLYMORPHISM
                    (POLYMORPHISM OR POLYMORPHISMS)
          242134 MUTATION
```

```
        158483 MUTATIONS
        301944 MUTATION
                 (MUTATION OR MUTATIONS)
         19356 ALLELIC
             1 ALLELICS
         19357 ALLELIC
                 (ALLELIC OR ALLELICS)
L9          40 L8 AND (POLYMORPHISM OR MUTATION OR ALLELIC)

=> s L9 AND (position 32 or 159 or 298 or 340 or 462)
        404805 POSITION
        167571 POSITIONS
        538168 POSITION
                 (POSITION OR POSITIONS)
        390931 32
           245 POSITION 32
                 (POSITION(W)32)
         27316 159
         46807 298
         40760 340
          5088 462
L10          0 L9 AND (POSITION 32 OR 159 OR 298 OR 340 OR 462)

=> s L9 AND residue
        377482 RESIDUE
        364163 RESIDUES
        656073 RESIDUE
                 (RESIDUE OR RESIDUES)
L11          1 L9 AND RESIDUE

=> d ti L11

L11  ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Mode of Binding of Methyl Acceptor Substrates to the Adrenaline-
     Synthesizing Enzyme Phenylethanolamine N-Methyltransferase: Implications
     for Catalysis


=> s L9 and variation
        393901 VARIATION
        260671 VARIATIONS
        614708 VARIATION
                 (VARIATION OR VARIATIONS)
L12          6 L9 AND VARIATION

=> d 1-6 ti,so,ibib,abs L12

L12  ANSWER 1 OF 6  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Human phenylethanolamine N-methyltransferase pharmacogenomics: Gene
     re-sequencing and functional genomics
SO   Journal of Neurochemistry (2005), 95(6), 1766-1776
     CODEN: JONRA9; ISSN: 0022-3042
ACCESSION NUMBER:         2006:14627  CAPLUS
DOCUMENT NUMBER:          144:248259
TITLE:                    Human phenylethanolamine N-methyltransferase
                          pharmacogenomics: Gene re-sequencing and functional
                          genomics
AUTHOR(S):                Ji, Yuan; Salavaggione, Oreste E.; Wang, Liewei;
                          Adjei, Araba A.; Eckloff, Bruce; Wieben, Eric D.;
                          Weinshilboum, Richard M.
CORPORATE SOURCE:         Departments of Molecular Pharmacology & Experimental
                          Therapeutics, Mayo Clinic College of Medicine,
                          Rochester, MN, USA
```

SOURCE: Journal of Neurochemistry (2005), 95(6), 1766-1776
CODEN: JONRA9; ISSN: 0022-3042
PUBLISHER: Blackwell Publishing Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Phenylethanolamine N-methyltransferase (PNMT, EC2.1.1.28) catalyzes the N-methylation of norepinephrine to form epinephrine. As a step toward understanding the possible contribution of inheritance to individual variation in PNMT-catalyzed epinephrine formation, were-sequenced the entire human PNMT gene, including the three exons, the introns and approx. 1 kb of the 5'-flanking region (5'-FR), using DNA samples from 60 African-American (AA) and 60 Caucasian-American (CA) subjects. Within the 3.5 kb re-sequenced, 18 single nucleotide polymorphisms (SNPs) were observed, including four non-synonymous coding SNPs (cSNPs) that resulted in the following alterations in encoded amino acid sequence; Asn9Ser, Thr98Ala, Arg112Cys and Ala175Thr. When constructs for the non-synonymous cSNPs were transiently expressed in COS-1 cells, the Ala98 allozyme displayed significantly lower levels of both activity and immunoreactive protein ($p < 0.002$) than did the wild-type (WT) enzyme due, at least in part, to accelerated protein degradation by a proteasome-mediated process. Luciferase reporter gene constructs were also created for the six common PNMT 5'-FR haplotypes observed Significant differences were observed among haplotypes in their ability to drive transcription. These observations raise the possibility of inherited variation in the ability to form epinephrine from norepinephrine as a result of variant PNMT polymorphisms and haplotypes.

REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN

TI Single nucleotide polymorphisms predictive for cardiovascular disease, adverse drug reactions, and drug efficacy

SO Eur. Pat. Appl., 383 pp.
CODEN: EPXXDW

ACCESSION NUMBER: 2004:181841 CAPLUS
DOCUMENT NUMBER: 140:230590
TITLE: Single nucleotide polymorphisms predictive for cardiovascular disease, adverse drug reactions, and drug efficacy
INVENTOR(S): Schwers, Stephan; Kallabis, Harald; Stropp, Udo
PATENT ASSIGNEE(S): Bayer Healthcare AG, Germany
SOURCE: Eur. Pat. Appl., 383 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1394267	A1	20040303	EP 2002-18158	20020819
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
WO 2004018709	A2	20040304	WO 2003-EP9126	20030818
WO 2004018709	A3	20041028		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,				

KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2003266291 A1 20040311 AU 2003-266291 20030818

EP 1532277 A2 20050525 EP 2003-792358 20030818

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

PRIORITY APPLN. INFO.: EP 2002-18158 A 20020819

WO 2003-EP9126 W 20030818

AB The invention provides diagnostic methods and kits including oligo and/or polynucleotides or derivs., including as well antibodies determining whether a human subject is at risk of getting adverse drug reaction after statin therapy or whether the human subject is a high or low responder or a good a or bad metabolizer of statins. The invention provides further diagnostic methods and kits including antibodies determining whether a human subject is at risk for a cardiovascular disease. Still further the invention provides polymorphic sequences and other genes. The present invention further relates to isolated polynucleotides encoding a phenotype associated (PA) gene polypeptide useful in methods to identify therapeutic agents and useful for preparation of a medicament to treat cardiovascular disease or influence drug response, the polynucleotide is selected from the group comprising: SEQ ID 1-168 with allelic variation as indicated in the sequences section contained in a functional surrounding like full length cDNA for PA gene polypeptide and with or without the PA gene promoter sequence.

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN

TI Phenylethanolamine N-methyltransferase G-148A genetic variant and weight loss in obese women

SO Obesity Research (2003), 11(3), 415-419
CODEN: OBREFR; ISSN: 1071-7323

ACCESSION NUMBER: 2003:246374 CAPLUS

DOCUMENT NUMBER: 138:383505

TITLE: Phenylethanolamine N-methyltransferase G-148A genetic variant and weight loss in obese women

AUTHOR(S): Peters, Warren R.; MacMurry, James P.; Walker, Jennifer; Giese, Russell J., Jr.; Comings, David E.

CORPORATE SOURCE: Center for Health Promotion, Loma Linda University, Loma Linda, CA, USA

SOURCE: Obesity Research (2003), 11(3), 415-419
CODEN: OBREFR; ISSN: 1071-7323

PUBLISHER: North American Association for the Study of Obesi ty

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Objective: To understand the impact of the phenylethanolamine N-methyltransferase (PNMT) G-148A gene and nutritional variables on weight loss in obese women. Research Methods and Procedures: One hundred forty-nine women, ages 45 to 65 with a body mass index of >30 kg/m2, participated in a 6-mo, open-label intervention that included sibutramine (15 mg/d) and a monthly health-education class. Anthropometric measurements, vital signs, food frequency, exercise log, medication compliance, and psychol. and sociol. questionnaires were completed each month. Genetic polymorphisms of PNMT were determined Results: Univariate anal. of G/G, G/A, and A/A genotypes against tertiles of percentage of weight loss were significant at 3 but not at 6 mo (Pearson X2 : $p < 0.006$; homozygous/heterozygosity: $p < 0.002$, $p < 0.253$, and $p < 0.122$, resp.). A regression model that included the PNMT genetic variation and certain nutrition and exercise variables demonstrated that only the PNMT gene ($\beta = 0.360$, SE 0.585, and $p = 0.003$) was statistically significant at 6 mo, and the total calories ($\beta = -0.925$, SE = 0.004, and $p = 0.009$), fiber intake ($\beta =$

0.621, SE = 0.124, and p = 0.000), and PNMT (β = 0.262, SE = 1.415, and p = 0.024) were significant. Discussion: The homozygosity/heterozygosity of the PNMT gene was highly predictive of significant weight loss with sibutramine during the first 3 mo, which highlights the need for specific pharmacotherapy. The early weight-loss success of those subjects who were homozygous for PNMT may have motivated and selected those that would make further dietary changes, which then augmented their final weight loss.

REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN

TI Identification of 197 genetic variations in six human methyltransferase genes in the Japanese population

SO Journal of Human Genetics (2001), 46(9), 529-537
CODEN: JHGEFR; ISSN: 1434-5161

ACCESSION NUMBER: 2001:706233 CAPLUS
DOCUMENT NUMBER: 136:351050
TITLE: Identification of 197 genetic variations in six human methyltransferase genes in the Japanese population
AUTHOR(S): Saito, Susumu; Iida, Aritoshi; Sekine, Akihiro; Miura, Yukie; Sakamoto, Tsutomu; Ogawa, Chie; Kawauchi, Saori; Higuchi, Shoko; Nakamura, Yusuke
CORPORATE SOURCE: Laboratory for Genotyping. SNP Research Center, Institute of Physical and Chemical Research, Tokyo, Japan
SOURCE: Journal of Human Genetics (2001), 46(9), 529-537
CODEN: JHGEFR; ISSN: 1434-5161
PUBLISHER: Springer-Verlag Tokyo
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Methylation is an important event in the biotransformation pathway for many drugs and xenobiotic compds. We screened DNA from 48 Japanese individuals for single-nucleotide polymorphisms (SNPs) in six methyltransferase (MT) genes (catechol-O-MT, COMT; guanidinoacetate N-MT, GAMT; histamine N-MT, HNMT; nicotinamide N-MT, NNMT; phosphatidylethanolamine N-MT, PEMT; and phenylethanolamine N-MT, PNMT) by direct sequencing of their entire genomic regions except for repetitive elements. This approach identified 190 SNPs and seven insertion/deletion polymorphisms among the six genes. Of the 190 SNPs, 33 were identified in the COMT gene, 6 in GAMT, 41 in HNMT, 8 in NNMT, 98 in PEMT, and 4 in PNMT. Nine were located in 5' flanking regions, 156 in introns, 10 in exons, and 15 in 3' flanking regions. These variants may contribute to a more precise understanding of possible correlations between genotypes and disease-susceptibility phenotypes or risk for side effects from drugs.

REFERENCE COUNT: 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN

TI Gene probes used for genetic profiling in healthcare screening and planning

SO PCT Int. Appl., 745 pp.
CODEN: PIXXD2

ACCESSION NUMBER: 1999:795994 CAPLUS
DOCUMENT NUMBER: 132:31744
TITLE: Gene probes used for genetic profiling in healthcare screening and planning
INVENTOR(S): Roberts, Gareth Wyn
PATENT ASSIGNEE(S): Genostic Pharma Ltd., UK
SOURCE: PCT Int. Appl., 745 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9964627	A2	19991216	WO 1999-GB1780	19990604

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:		
GB 1998-12099	A	19980606
GB 1998-13291	A	19980620
GB 1998-13611	A	19980624
GB 1998-13835	A	19980627
GB 1998-14110	A	19980701
GB 1998-14580	A	19980707
GB 1998-15438	A	19980716
GB 1998-15574	A	19980718
GB 1998-15576	A	19980718
GB 1998-16085	A	19980724
GB 1998-16086	A	19980724
GB 1998-16921	A	19980805
GB 1998-17097	A	19980807
GB 1998-17200	A	19980808
GB 1998-17632	A	19980814
GB 1998-17943	A	19980819

AB There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response. In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol. states of interest. According to the invention, the number of genes and their configurations (mutations and polymorphisms) needed to be identified in order to provide critical clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies which comprises of the identification of the core group of genes and their sequence variants required to provide a broad base of clin. prognostic information - "genostics". The "Genostic" profiling of patients and persons will radically enhance the ability of clinicians, healthcare professionals and other parties to plan and manage healthcare provision and the targeting of appropriate healthcare resources to those deemed most in need. The use of this invention could also lead to a host of new applications for such profiling technologies, such as identification of persons with particular work or environment related risk, selection of applicants for employment, training or specific opportunities or for the enhancing of the planning and organization of health services, education services and social services.

L12 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN

TI Gene probes used for genetic profiling in healthcare screening and planning
SO PCT Int. Appl., 149 pp.
CODEN: PIXXD2

ACCESSION NUMBER: 1999:795993 CAPLUS
DOCUMENT NUMBER: 132:31743
TITLE: Gene probes used for genetic profiling in healthcare screening and planning
INVENTOR(S): Roberts, Gareth Wyn
PATENT ASSIGNEE(S): Genostic Pharma Limited, UK
SOURCE: PCT Int. Appl., 149 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9964626	A2	19991216	WO 1999-GB1779	19990604
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2330929	AA	19991216	CA 1999-2330929	19990604
AU 9941586	A1	19991230	AU 1999-41586	19990604
AU 766544	B2	20031016		
AU 9941587	A1	19991230	AU 1999-41587	19990604
GB 2339200	A1	20000119	GB 1999-12914	19990604
GB 2339200	B2	20010912		
EP 1084273	A1	20010321	EP 1999-925207	19990604
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2003528564	T2	20030930	JP 2000-553616	19990604
US 2003198970	A1	20031023	US 2002-206568	20020729

PRIORITY APPLN. INFO.:

GB 1998-12098	A	19980606
GB 1998-28289	A	19981223
GB 1998-16086	A	19980724
GB 1998-16921	A	19980805
GB 1998-17097	A	19980807
GB 1998-17200	A	19980808
GB 1998-17632	A	19980814
GB 1998-17943	A	19980819
US 1999-325123	B1	19990603
WO 1999-GB1779	W	19990604

AB There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response. In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol. states of interest. According to the invention, the number of genes and their configurations (mutations and polymorphisms) needed to be identified in order to provide

critical clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies.

=>